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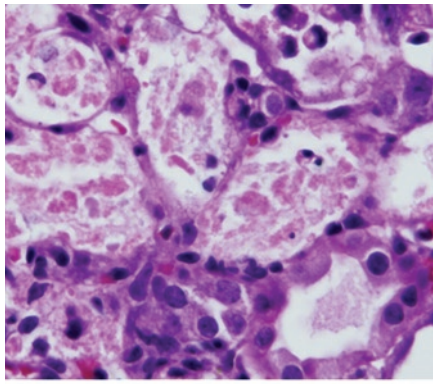
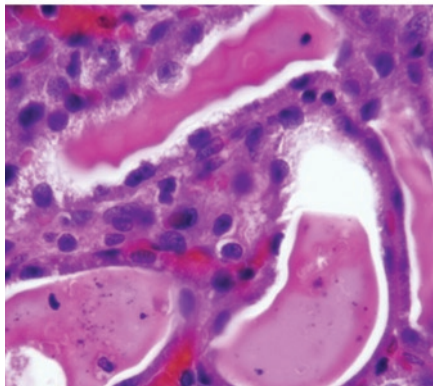
Pyrophosphate and vascular calcification

Vascular calcification is emerging as an important risk factor for cardiovascular mortality in end-stage renal disease (ESRD). Given the role that high phosphate levels in ESRD have in promoting calcification, efforts to find agents that reverse or prevent calcification have received a high priority in research. As they report in this issue, O'Neill *et al.* used pyrophosphate as a potential therapeutic agent in experimental renal failure. They produced uremia in rats, fed them a high-phosphate diet, and then injected some of them with pyrophosphate. The incidence of calcification in the aorta remained the same, but their calcium content was drastically reduced by 70% with no effect on bone formation. These studies provide a new potential treatment for this problem. **See page 512.**

Biomarkers for acute kidney injury due to toxins

Although the diagnosis of acute renal failure is usually straightforward, the identification of the cause is often challenging. In this issue, two papers attempt to provide new diagnostic tools that might differentiate one cause from another. Ferreira *et al.* administered toxic doses of gentamicin or cisplatin to rats and examined their urine





by two-dimensional gel electrophoresis, followed by sequencing of appropriate peptides. They found that the rats with gentamicin nephrotoxicity, but not those with cisplatin toxicity, had elevated levels of regenerating islet-derived protein III β (reg IIIb) and gelsolin in their urine. In another study, Kwon *et al.* followed up on the observation that not every animal given cisplatin develops renal failure. They used nuclear magnetic resonance spectroscopy to compare the excretion of metabolites in the urine of those that developed renal failure versus those that did not. A number of compounds were found that together were able to predict the sensitive animals. However, the application of these findings to the clinical setting has yet to occur. **See page 518 and page 529.**

Predicting the response to erythropoietin

Patients with ESRD who receive erythropoietin vary in their response, so it is important to identify those who will respond before treatment. Clinically, parameters of iron metabolism need to be examined first, because they may be the culprit in the response. However, there is still a need to distinguish the responders from the non-responders. As they report in this issue, Merchant *et al.* examined the sera of 35 patients by mass spectrometry and identified 91 peptides, 16 of which differed in abundance between good and poor responders. Three of these peptides, derived from oncostatin receptor β, were highly associated with poor response, although fibrinogen, factor XIII, complement factor 3, and cysteine/histidine-rich 1 protein (CYHR1) were higher in good responders. Western blots confirmed and extended these studies. Hence, two new biomarkers are presented that should prove useful in difficult cases in which prediction of the response to erythropoietin is needed. **See page 546.**